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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/576,274	10/09/2007	Rehab Al-Jamal	MUR-06-1101	9435

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IP GROUP OF DLA PIPER LLP (US)
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EXAMINER	
HADDAD, MAHER M	

ART UNIT	PAPER NUMBER
1644	

NOTIFICATION DATE	DELIVERY MODE
05/28/2010	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

pto.phil@dlapiper.com

Office Action Summary	Application No.	Applicant(s)	
	10/576,274	AL-JAMAL ET AL.	
	Examiner	Art Unit	
	Maher M. Haddad	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *26 April 2010*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,4,16,20-29 and 31-34* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,4,16,20-29,31 and 32* is/are rejected.

7)☒ Claim(s) *33-34* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

RESPONSE TO APPLICANT'S AMENDMENT

1. Applicant's amendment, filed 04/26/2010, is acknowledged.

2. Claims 1, 4, 16, 20-29 and 31-34 are pending and under examination in the instant application.

3. In view of the amendment filed on 04/26/2010, only the following rejections are remained.

4. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

5. Claims 1, 4, 16, 20-29 stand and newly added claims 31-32 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention for the same reasons set forth in the previous Office Actions.

Applicant is in possession of a method of promoting tissue repair in lung emphysema comprising administering an antibody that binds to the beta 1 integrin molecule in a region of amino acid residue 82-87 comprising residues TAEKLK (SEQ ID NO:1) of the sequence of the mature beta 1 integrin molecule.

Applicant is not in possession of the methods recited in claims 1, 4, 16, 20-29 and 31-34.

The claims recite the anti-TAEKLK antibody and functionally modulation of beta 1 integrin results in "an alteration in the metalloproteinase balance" as part of the invention.

The specification on ¶ 17, discloses that recent reports have demonstrated that β1 integrin can also bind metalloproteinases such as MMP2 and MMP9 and affect their activation state. Both MMPs have been shown to contribute to caspase-mediated brain endothelial cell death after hypoxia-reoxygenation by disrupting cell-matrix interactions and homeostatic integrin signalling. TGFβ1 have also been reported to bind to β1 integrin. The specification on ¶136 discloses that the ECM response to β1 integrin functional modification was accompanied by a decrease in cell death and increase in TIMP1, inactive MP9 and active TGFβ1 and a decrease in MMP1. However, it is not clear from the specification whether (1) the binding between β1/MMPs occurs via the claimed TAEKLK residues and (2) the claimed antibodies would act as metalloproteinase (MMP) inhibitors and/or activator in order to promote tissue repair, wherein extracellular matrix of the tissue has been degraded. The specification does not provide a genus of anti-β1 antibodies that binds TAEKLK which would function in the alteration/modulation in the metalloproteinase balance. The specification fails to disclose that binding of β1 integrin via TAEKLK to MMP2

and MMP9 leads to their activation or inhibition. The specification fails to show that anti-TAEKLK antibodies lead to modulation in the metalloproteinase. CORRY et al (*The FASEB Journal.* 2004;18:995-997) teach that similar to MMP2-deficient mice, WT, $MMP9^{-/-}$, and MMP9/MMP2 double knockout (dko) mice immunized with CAA showed characteristic allergic and obstructive features, including similar degrees of airway hyperresponsiveness, mucin, and glycoprotein hypersecretion and elevated serum IgE levels (see page 995, under section 2). Accordingly, inactivation of MMP9 with claimed antibodies lead to airway hyperresponsiveness, mucin, and glycoprotein hypersecretion and elevated serum IgE levels.

There does not appear to be an adequate written description in the specification as-filed of the essential structural feature that provides the recited function of modulation in the metalloproteinase balance that would lead to the promotion of tissue repair. The Guidelines for the Examination of Patent Applications Under the 35 U.S.C. 112, ¶ 1 "Written Description" Requirement make clear that the written description requirement for a claimed genus may be satisfied through sufficient description of a representative number of species by actual reduction to practice, reduction to drawings, or by disclosure of relevant, identifying characteristics, i.e., structure or other physical and or chemical properties, by functional characteristics coupled with a known or disclosed correlation between function and structure, or by a combination of such identifying characteristics, sufficient to show the applicant was in possession of the genus (Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 2001, see especially page 1106 3rd column).

Applicant's arguments, filed 04/26/2010, have been fully considered, but have not been found convincing.

Applicant points to well known clone that binds to the same sequence 82-87 (TAEKLK, SEQ ID NO:1) of β1 integrin in the art such as SG/7, SG/19, C30B and D11B. However, none of the taught clones are shown to cause alteration in the metalloproteinase balance.

6. Claims 1, 4, 16, 20-29 stand and newly added claims 31-32 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of promoting tissue repair in lung emphysema comprising administering antibodies that binds TAEJKJ of SEQ ID NO:1, does not reasonably provide enablement for methods claimed in claims 1, 4, 16, 20-29 and 31-34. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims for the same reason set forth in the previous Office Actions.

Applicant's arguments, filed 04/26/2010, have been fully considered, but have not been found convincing.

Applicant submits that an alteration in the metalloproteinase balance refers to an increase in certain metalloproteinases and a decrease in other metalloproteinases, for example, an increase in

inactive MMP9 and a decrease in MMP1. Applicant concluded that the skilled practitioner would have a reasonable expectation that the same compounds would achieve such an alteration.

However, the claims are not limited to MMP9 and MMP1. Further, CORRY et al (*The FASEB Journal.* 2004;18:995-997) teach that similar to MMP2-deficient mice, WT, $MMP9^{-/-}$, and MMP9/MMP2 double knockout (dko) mice immunized with CAA showed characteristic allergic and obstructive features, including similar degrees of airway hyperresponsiveness, mucin, and glycoprotein hypersecretion and elevated serum IgE levels (see page 995, under section 2). Accordingly, inactivation of MMP9 with claimed antibodies lead to airway hyperresponsiveness, mucin, and glycoprotein hypersecretion and elevated serum IgE levels. Moreover, the skilled in the art would not know which MMP is involved in promoting tissue repair, wherein extracellular matrix of tissue has been degraded and whether an increase or decrease in the specific MMP would result in modulation in the metalloproteinase balance that would be beneficial to promote tissue repair wherein extracellular matrix of the tissue has been degraded.

Applicant points that working examples have been provided for emphysema, Parkinson's disease, arthritis and Alzheimer's to support the full scope of any and every tissue repair and tissue injury.

However, besides emphysema, the specification fails to contemplate treating Parkinson's disease, arthritis or Alzheimer's with the claimed antibodies. Therefore, the Examiner cannot enable Applicant for such disease (i.e., creates new matter issue). Moreover, emphysema, Parkinson's disease, arthritis and Alzheimer's are not representative of the whole genus of tissue repair wherein extracellular matrix of the tissue has been degraded. For example, said tissue repair encompasses skin tissue, tissue of the central nervous system, liver tissue, kidney tissue, the cardiovascular system, bone tissue and cartilage (see instant claim 31). Accordingly, Applicant's examples are insufficient to show the claimed methods can extrapolated to the claimed types of tissue repair and tissue injury wherein the extracellular matrix is degraded.

7. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

8. Claims 1, 4, 16, 20-25, 28-29 stand and newly added 31-32 are rejected under 35 U.S.C. 102(b) as being anticipated by US20030109435, as is evidenced by Al-Jamal's declaration, filed 12/28/2009 and Chemicon International catalog no. MAB1965, 9/23/09 (submitted by Applicant on 06/03/2009) for the reasons of record.

Applicant's arguments, filed 04/26/2010, have been fully considered, but have not been found convincing.

Applicant submits that the `435 publication fails to teach all of the elements of the claimed invention. The `435 publication does not disclose, teach or suggest the use of the JB1a (or other antibodies which bind the same sequences) for promoting tissue repair or treating tissue injury where the extracellualr matrix is degraded. Applicant also submits that the `435 publication does not disclose, teaextracellular that specifically targeting the hybrid domain of beta 1 integrin modulates function of beta 1 integrin, nor that targeting the hybrid domain has agonist/antagonist properties (i.e., functional modulation or conformational modulation). Instead, it teaches that inhibition of beta1 integrin using AIIIB2 and JB1a amongst other anti-integrin antibodies inhibited neuronal toxicity by inhibiting the formation of amyloid fibrils. Applicant submits that the `435 enlists that tocicity of preformed amyloid fibrils is only evident when added in conjunction with soluble amyloid. The reason given is that the presence of soluble amyloid enhances the cells' ability to form amyloid fibrils. In the Applicant's working examples, no soluble amyloid was added and, as such, targeting the hybrid domain using JB1a could not have inhibited amyloid fibril formation as those fibrils were preformed separately prior to the study, as nomally performed by those skilled in the art. There is no disclosure in the US `435 that targeting beta 1 integrin's hybrid domain could induce cellular repair. Applicant concluded that the `435, no neurotoxicity was observed within neuronal cultures were treated with amyloid fibrils alone. In addition, Applicant submits that upon examination of the staining pictures of the neurons provided in the `435, the cells appear dedifferentiated and, as such, these cells do not behave like a normal cerebral neuron with neurite extension and a distinct cell body. Applicant submits that all studies examining effects on primary neurons entail pretreatment with growth factors and activators (NGF, IBMX, TPA) to revert to the neuronal phenotype cell morphology. Applicant concluded that the cells shown in the examples are not typical neurons and those skilled in the relevant art would not expect to extrapolate any findings to in vivo studies.

However, preamble language in claims of patents directed to promoting tissue repair wherein extracellular matrix of the tissue has been degraded are expressions of purposes and intended results, and as such are non-limiting, since language does not result in manipulative difference in steps of claims. In re Hirao 190 USPQ 15, 16-17, (CCPA 1976) held that the preamble was non-limiting because it merely recited the purpose of the process, which was fully set forth in the body of the claim. With respect to the soluble amyloid, the Examiner notes that the instant claims recite the term "comprising" administering an antibody, which is open-ended. It would open up the claim to include the soluble amyloid. Moreover, the `435 publication is not limited of the addition of soluble amyloid, the `435 publication claims treating Parkinson's disease (see published claims 43 and 92) with an antibody that recognizes the same epitope (82-87 of β1) as antibody MAB 1965 (claimed clone JB1a) (see published claims 1, 4, 14, 21-39, 50, 53, 70-88). Regarding, the dedifferentiated neurons, it appears that applicant relies upon certain mechanism of action, cell morphology and neuronal phenotype but does not provide objective evidence that the prior art teaching of treating the same Parkinson's patient populations with the same compositions to achieve the same therapeutic effect differs from the claimed methods.

The claimed functional limitations would be inherent properties of the clone JB1a.

9. Claims 1, 4, 16, 19, 21-25 and 28-29 stand rejected under 35 U.S.C. 102(b) as being anticipated by US. Pat. 6,123,941 for the reasons of record.

Applicant's arguments, filed 04/26/2010, have been fully considered, but have not been found convincing.

Applicant argues that the `941 fails to teach all of the elements of the claims. It does not disclose, teach or suggest the use of the JB1a antibody (or other antibodies which bind the same sequences) for promoting tissue repair or treating tissue injury wherein the extracellular matrix is degraded. The US `941, tumor cells were able to synthesize extracellular matrix, albeit disorganized. US `941 claims that targeting the beta integrin using AIIB2 enhances normal organization of the matrix leading to an alteration in cell behavior wherein cells revert from a malignant phenotype to a normal phenotype. The Applicants' claimed compounds are used to promote tissue repair or treat tissue injury where the extracellular matrix is degraded. The Applicants submit that degradation is higher than synthesis and that use to enhance matrix organization differs from use to promote tissue repair or treat tissue injury where the extracellular matrix is degraded. Applicant further submits that the work of Bissel et al mainly details the effect of functional inhibition of beta1 integrin in mammary tumor cells in vitro using the clone AllB2, which binds beta A domain within amino acids 207-218. This was not clearly discerned in the patent but is in the research publications of the data detailed in the patent. US `941 states at col., 5, line 64 with respect to the use of AIIB2 that "note that T4 cells revert to normal phenotype when beta1 integrin function-blocking antibody (Ab) is applied but that "normal cells die as a result of application of the beta integrin function-blocking antibody". Us `941 therefore teaches that using AIIB2 on non-transformed or non-cancerous cells would in fact increase cell death. The pro-apoptotic effect of AIIB2 on normal human cells would cause serious adverse side effects. This effect could not have been seen in the in vivo experiments in mice as AIIB2 was not shown to cross react with mouse beta1 integrin. The cells to be treated by the Applicants are non-tumor cells which are dying as a result of injury. Furthermore, in the Applicants' emphysema model, there was no histological evidence of proliferation associated with injury or its reversal on the basis of the morphology in the H&E sections used in the patent (Ki67 staining was done on lung section). Targeting the hybrid domain of beta1 integrin using JB1a, unlike the AIIB2 listed in US`941, prevented apoptosis and has not demonstrated any detectable side effects of such severity in both in vitro and in vivo studies.

However, preamble language in claims of patents directed to promoting tissue repair wherein extracellular matrix of the tissue has been degraded are expressions of purposes and intended results, and as such are non-limiting, since language does not result in manipulative difference in steps of claims. In re Hirao 190 USPQ 15, 16-17, (CCPA 1976) held that the preamble was non-limiting because it merely recited the purpose of the process, which was fully set forth in the body of the claim. The `941 patent teaches that T4 cells revert to normal phenotype when β1 integrin function-blocking antibody (Ab) is applied but that normal cells die as a result of application of the β1 integrin function-blocking antibody. The level of response varies as a function of the concentration of applied Ab. It is important to use the correct concentration of β1

function-blocking Ab to balance these two effects (see col., 5&6 bridging ¶). It is not clear whether Applicant took into consideration the concentration of the antibody in emphysema model. There is only one concentration of 50 ug/animal. Applicant's claims are not limited to non-tumor cells as argued, but encompass any tissue injury including treating tumor. When claim 1 is given its broadest reasonable interpretation: promoting tissue repair can include reversing malignant phenotype in tissue as a result of an inhibition of the apoptotic pathway. The Examiner direct Applicant's attention to the instant specification on page 15, lines 2-21.

10. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> *(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.*

11. Claims 1 and 20 stand rejected under 35 U.S.C. 103(a) as being unpatentable over US. Pat. 6,123,941 as in view of Owens *et al* (1994) for the reasons of record.

Applicant's arguments, filed 04/26/2010, have been fully considered, but have not been found convincing.

Applicant submtis that Owens et al does not remedy these deficiencies.

It is the Examiner's position that claims 1 and 20 are obvious over the combined reference teachings.

12. Claims 1, 4, 15-16 and 19-30 are directed to an invention not patentably distinct from claims 1, 2, 5, 11, 16, 19, 24, 25, 32, 35, 57 and 59-63 of commonly assigned 12528749. Specifically, both applications are using the same antibody clone JB1a that binds amino acid residues 82-84 and possible 179-184 (as is evidenced by Al-Jamal and Harrison, Pharmacology & Therapeutics 120 (2008) 81-101, see Table 1) to teat tissue damage.

13. The U.S. Patent and Trademark Office normally will not institute an interference between applications or a patent and an application of common ownership (see MPEP Chapter 2300). Commonly assigned 12528749, discussed above, would form the basis for a rejection of the noted claims under 35 U.S.C. 103(a) if the commonly assigned case qualifies as prior art under 35 U.S.C. 102(e), (f) or (g) and the conflicting inventions were not commonly owned at the time the invention in this application was made. In order for the examiner to resolve this issue, the assignee can, under 35 U.S.C. 103(c) and 37 CFR 1.78(c), either show that the conflicting inventions were commonly owned at the time the invention in this application was made, or name the prior inventor of the conflicting subject matter.

A showing that the inventions were commonly owned at the time the invention in this application was made will preclude a rejection under 35 U.S.C. 103(a) based upon the commonly

assigned case as a reference under 35 U.S.C. 102(f) or (g), or 35 U.S.C. 102(e) for applications pending on or after December 10, 2004.

14. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

15. Claims 1, 4, 15-16 and 19-29 and 31-34 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 1, 2, 5, 11, 16, 19, 24, 25, 32, 35, 57 and 59-63 of copending Application No. 12528749. Although the conflicting claims are not identical, they are not patentably distinct from each other because both applications are using the same antibody clone JB1a that binds amino acid residues 82-84 and possible 179-184 (as is evidenced by Al-Jamal and Harrison, Pharmacology & Therapeutics 120 (2008) 81-101, see Table 1) to teat tissue damage.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

Applicant's arguments, filed 04/26/2010, have been fully considered, but have not been found convincing.

Applicants submit that they will address the rejection when it becomes non-provisional.

The rejection is maintained for reasons of record.

16. Claims 33-34 are objected to as being dependent upon a rejected base claim, but would be allowable if rewritten in independent form including the limitation of claim 16 and overcome the ODP rejection.

17. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

18. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Maher Haddad whose telephone number is (571) 272-0845. The examiner can normally be reached Monday through Friday from 7:30 am to 4:00 pm. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

May 24, 2010

/Maher M. Haddad/
Primary Examiner,
Art Unit 1644